AMERICAN JOURNAL OF EPIDEMIOLOGY
Vol. 123, No. 2
Printed in U.S.A.

ASSOCIATIONS BETWEEN SEVERAL SITES OF CANCER AND NINE ORGANIC DUSTS: RESULTS FROM AN HYPOTHESIS-GENERATING CASE-CONTROL STUDY IN MONTREAL, 1979–1983

JACK SIEMIATYCKI,[1,2] LESLEY RICHARDSON,[1] MICHEL GÉRIN,[3] MARK GOLDBERG,[1,2] RON DEWAR,[1,2] MARIE DÉSY,[1] SALLY CAMPBELL,[1] AND SHOLOM WACHOLDER[2]

Siemiatycki, J. (Institut Armand-Frappier, Laval-des-Rapides, Quebec, Canada, H7V 1B7), L. Richardson, M. Gérin, M. Goldberg, R. Dewar, M. Désy, S. Campbell, and S. Wacholder. Associations between several sites of cancer and nine organic dusts: results from an hypothesis-generating case-control study in Montreal, 1979–1983. *Am J Epidemiol* 1986;123:235–49.

A multi-cancer site, multi-factor case-control study was undertaken to generate hypotheses about possible occupational carcinogens. Probing interviews were carried out with over 2,000 subjects. All incident cases of 19 sites of cancer in males aged 35–70 and resident in Montreal were eligible. The interview was designed to obtain detailed lifetime job histories, and information on potential confounders. Each job history was reviewed by a team of chemists who translated it into a history of occupational exposures. These occupational exposures were then analyzed as potential risk factors in relation to the sites of cancer included. For each site of cancer analyzed as a case series, controls were selected from among the other cancer sites in the study. This report concerns the associations between sites of cancer for which there were over 100 cases processed (stomach; colorectal, also analyzed by subsites; lung; prostate; bladder; kidney; non-Hodgkin's lymphoma) and nine organic dusts (wood; paper; grain; flour; fabrics; cotton; wool; synthetics; fur). All site-exposure combinations were investigated. The ones that provided the most interesting leads were lung-wood dust (odds ratio (OR) = 1.5), stomach-wood dust (OR = 1.5), colorectal-synthetic fiber (OR = 1.5), bladder-synthetic fiber (OR = 1.8), non-Hodgkin's lymphoma-cotton dust (OR = 1.9), colon-grain dust (OR = 2.6), prostate-grain dust (OR = 2.2), and prostate-paper dust (OR = 2.0). Only the associations with wood dust, synthetic fibers and cotton dust showed some evidence of "dose-response" with duration of exposure. Because it is such a common exposure and appears to increase lung and stomach cancer risks, wood dust may be responsible for a great deal of occupational cancer.

dust; lung neoplasms; neoplasms; occupational diseases; textile industry; textiles; wood

Of the thousands of substances to which workers are exposed in the workplace, only a handful have ever been assessed in epidemiologic studies for carcinogenic poten-

Received for publication March 26, 1985, and in final form July 22, 1985.

Abbreviation: ICD, *International Classification of Diseases*.

[1] Centre de Recherche en Épidémiologie et Médecine Préventive, Institut Armand-Frappier, 531 boulevard des Prairies, Laval-des-Rapides, Québec, Canada, H7V 1B7. (Reprint requests to Dr. Jack Siemiatycki.)

[2] Department of Epidemiology and Biostatistics, McGill University, Montreal.

[3] Département de Médecine du Travail et d'Hygiène du Milieu, Université de Montréal, Montréal, Canada.

This research was supported by grants from the Institut de Recherche en Santé et Sécurité du Travail du Québec, the National Health Research and Development Program, and the National Cancer Institute of Canada.

tial (1). Most known human carcinogens were discovered by clinicians as the result of a series of coincidences. The tumor type had to be unusual to be noticed; the relative risk had to be quite high to induce a remarkable number of cases in a workforce; the carcinogen had to affect a workforce whose common bond was evident (e.g., they worked in a single plant or had a common occupation); the cluster of cases had to come to the attention of a single clinician who was persistent enough to follow up with a report of the event. In the absence of any of these characteristics the hazard would not likely be discovered. Since it takes such a series of coincidences, it is reasonable to suspect that many occupational carcinogens have not yet been discovered.

Previously we argued that some systematic monitoring procedure was needed to generate hypotheses (2). We outlined an approach consisting of a population-based case-referent study including many sites of cancer and based on an attempt to obtain fairly detailed lifetime job histories from subjects, and to translate each job history into a history of occupational exposures. The occupational exposures thus inferred become factors for analysis in relation to cancer. The attempt to translate jobs into occupational exposures is similar in principle to attempts by Hoar et al. (3) and Coggon et al. (4) to use job-exposure matrices. However, our approach differs in the crucial respect that rather than having a predefined set of exposures that are automatically assigned to a given job category, we have a team of chemists and hygienists who consider the details and idiosyncracies of each job description and assign exposures in accordance with all available information about each unique situation.

Nineteen sites of cancer are included in the study; for each patient, information is obtained concerning past exposure to about 300 substances. The overall strategy is periodically to analyze subsets of the data to determine whether there seem to be any remarkable cancer-exposure associations.

This report concerns nine sites of cancer for which at least 100 subjects have been interviewed and coded—namely, stomach, colon except sigmoid, sigmoid and rectosigmoid junction, rectum, lung, prostate, bladder, kidney, and non-Hodgkin's lymphoma. The cutoff of at least 100 subjects was based on statistical power considerations. The risk factors under investigation are the following nine organic dusts: wood, paper, fabrics, wool, synthetic fibers, cotton, fur, grain, and flour. We wanted to examine a group of exposures that have relatively similar chemical/physical characteristics. It was also desirable to analyze common occupational exposures; and, in fact, the industries in which these exposures are found—forestry, construction, textile, fur, and food handling—are among the major industries in the Province of Quebec.

Several of the nine substances are naturally occurring and are among the most

Chemical coding of jobs was carried out by Howard Kemper, Denis Bégin, Ramzan Lakhani, Louise Nadon, and Lucien Laroche. Case ascertainment and interviewing were carried out by Denise Bourbonnais, Yves Céré, Lucy Felicissimo, Hélène Sheppard, Vincent Varacalli, and Michel Vinet. Jean Pellerin helped with data management. Claudette Richer prepared the manuscript. This study would not have been possible without the cooperation of the following clinicians and pathologists: Drs. Y. Méthot, R. Vauclair and Y. Ayoub—Hôpital Notre-Dame; Dr. R. Hand—Royal Victoria Hospital; Drs. C. Lachance and H. Frank—Sir Mortimer B. Davis Jewish General Hospital; Drs. W. P. Duguid and J. MacFarlane—Montreal General Hospital; Drs. S. Tange and D. Munro—Montreal Chest Hospital; Drs. F. Gomes and F. Wiegand—Queen Elizabeth Hospital; Drs. B. Artenian and G. Pearl—Reddy Memorial Hospital; Drs. D. Kahn and C. Pick—St. Mary's Hospital; Dr. C. Piché—Hôpital Ste-Jeanne d'Arc; Drs. P. Bluteau and G. Arjane—Centre Hospitalier de Verdun; Drs. Y. McKay and A. Bachand—Hôpital du Sacré-Coeur; Drs. A. Neaga and A. Reeves—Hôpital Jean-Talon; Drs. Y. Boivin and M. Cadotte—Hôtel-Dieu de Montréal; Dr. A. Iorizzo—Hôpital Santa Cabrini; Dr. A. Bonin—Hôpital Fleury; Drs. J. Lamarche and G. Lachance—Hôpital Maisonneuve-Rosemont; Drs. G. Gariépy and S. Legault-Poisson—Hôpital St-Luc; Dr. M. Mandavia—Lakeshore General Hospital; Dr. J. C. Larose—Cité de la Santé. The authors thank the pathology department and tumor registry staff of above-mentioned hospitals who notify them of new cases. They further thank Drs. Duncan Thomas, Ben Armstrong, and Nereo Segnan for comments on the manuscript and on various statistical issues.

common and oldest of occupational exposures. These substances are not completely distinct one from another. Fabric dust is a general term that refers to finished textile products and may involve exposure to cotton, wool or synthetic fibers. The latter exposures may occur in textile production; fabric dust would not. (The terms "dust" and "fiber" are used in their commonly understood sense, not according to any strict technical definitions.)

Aside from sinonasal cancer among wood-exposed furniture workers (5), there are no well-documented cancer risks due to these organic dusts. However, this reflects lack of investigation more than it does evidence of no effect. This report will present results on each of the cancer-exposure associations, focusing in particular on those that seem to be remarkable.

Methods

Multi-exposure multi-site monitoring study

The design described briefly here was detailed elsewhere (2, 6, 7). Nineteen sites of cancer were selected for study among males aged 35–70, resident in the area of Montreal. Case ascertainment procedures were established in the 19 major hospitals, allowing us access to nearly all incident cases in the target population. Diagnoses had to be histologically confirmed for a case to be eligible.

The cases available for this analysis were incident from September 1979 to June 1983. However, we were not ascertaining all sites of cancer without interruption during this period. Because of limited resources there were periods when we were not ascertaining one or more of the sites. Such ascertainment gaps were in effect in all hospitals. Each summer, for all sites, there was a two- to four-month ascertainment gap, depending on the interviewers' backlog from the previous year. Lung cancer was excluded in the second and third years. Rectal cancer was excluded in the first and second year.

Active regular contact with hospital pathology departments provided rapid case notification. In the period covered by this report, 2,610 subjects were eligible for inclusion in the study. An interviewer visited the patient in hospital or at home, as required. Approximately half the cases were still in hospital when first contacted by the interviewer and the rest had been discharged or were diagnosed as outpatients. Completed interviews or questionnaires were obtained from 2,180 subjects (83.5 per cent). Reasons for nonresponse were: refusals, 9.2 per cent; patient died, no next of kin found, 3.8 per cent; patient discharged, no valid address available, 3.6 per cent. Among the nine sites which had over 100 eligible cases, response rates varied from 81.9 per cent to 89.1 per cent, with most between 82 and 85 per cent. There was no evidence of nonresponse bias with respect to several variables which were available from medical records (8). Face-to-face interview with the subject was the strategy of choice and 82 per cent of completions were obtained this way. However, telephone interviews and specially designed self-administered forms were used for hard-to-interview subjects and these media provided 10 and 8 per cent, respectively, of completions. Proxy information was sought from next-of-kin for subjects who had died; 19 per cent of completions were obtained from next-of-kin.

The questionnaire was in two parts: a) a structured section requesting information on important potential confounders (e.g., ethnic group; residential history; schooling; home environment such as public water supply, home heating and cooking facilities; socioeconomic status currently and in childhood; consumption of cigarettes, alcohol, coffee, tea; hobbies; medical history; consumption of carotene-containing foods; height and weight), and b) a semistructured probing section designed to obtain a detailed description of each job the subject has had in his working lifetime. The interviewers were trained to probe for as much information as the patients can supply on the company's activities, the raw materials, final product, machines used, the man's

responsibilities for machine maintainance, the type of room or building in which he worked, activities of workmates around him, presence of gases, fumes or dusts, and any other information which could furnish a clue as to possible chemical or physical exposures incurred by the subject.

A team of chemists and hygienists working with us has had the responsibility of examining each completed questionnaire and translating each job into a list of potential exposures. They do this on a checklist form which explicitly lists some 300 of the most common occupational exposures. (See reference 7 for a description of the checklist and how the coding is performed.) For each product thought to be present in each job, the chemists noted their confidence that the exposure actually occurred (possible, probable, definite), frequency of exposure during a normal workweek (less than 5 per cent, 5–30 per cent, 30+ per cent) and concentration of the agent in the environment (low, medium, high). The concentration attribution is on a relative rather than absolute scale. The dates of beginning and ending of each job were recorded, and thus of the corresponding exposures in each job. The jobs themselves and the industries were coded according to standard Canadian classifications.

The team of chemists relied on the following sources as a basis for this retrospective exposure assessment: their own industrial experience and chemical knowledge, old and new technical and bibliographic material describing industrial processes, and consultations with experts familiar with particular industries. Neither interviewers nor chemists were aware of the patient's medical condition, eliminating one potential source of bias.

Case groups and control groups

The monitoring study design calls for periodic analyses on cumulated subjects interviewed. Each series of subjects with a common tumor is compared with one or more referent series drawn from the other cancer sites included in the study. This strategy has been discussed elsewhere (2).

For this report each case series consists of a topographically homogeneous entity with all histologic types combined. For the most part we used the three-digit level of the *International Classification of Diseases* (ICD) to define case series. However, there is some difference of opinion as to how colorectal cancers should be grouped for analysis. We analyzed as a case series the entire class of colorectal cancer as well as each of the three subcategories: colon (excluding sigmoid), sigmoid colon plus rectosigmoid junction, and rectum.

For each case series the control group consisted of all other sites in the study, with the following exceptions. Lung cancer was excluded from all control series. This was done because the very strong association between lung cancer and cigarette smoking is difficult to adjust for statistically; errors in measurement or in the functional form of the adjustment procedure could result in residual confounding. Also, for the two sites which were not ascertained in certain years of the study—lung and rectum—the controls consisted of other subjects who were ascertained in the same years as the corresponding cases. This was done on the chance that there was some variation in quality of interviewing or exposure assessment across the years of the study, despite our efforts to ensure consistency.

Table 1 shows the numbers of subjects interviewed and coded for each cancer site. For each of the sites which were analyzed as case series, the table shows the sites that served as controls. The table also shows for each site the number of controls thus selected and the resultant statistical power available to pick up an excess risk under two conditions: a twofold risk with a carcinogen to which 3 per cent of the workforce has been exposed, and a twofold risk with a carcinogen to which 10 per cent of the workforce has been exposed. For any carcinogen with over 10 per cent prevalence

TABLE 1

Sites of cancer with indications of numbers of cases, "control" sites† for each case series, numbers of controls and power to detect a twofold risk at two levels of prevalence of the carcinogen: study of associations of several cancer sites with nine organic dusts, Montreal, 1979–1983*

Site (ICD code)	Years (in the 4-year study period) in which the site was included	No. of cases	No. of controls	Power to detect 2-fold risk with exposure prevalence‡	
				3%	10%
Stomach (151)	1–4	156	1,524	0.60	0.93
Colon (153, except 153.3)	1–4	215	1,465	0.69	0.97
Rectosigmoid (153.3, 154.0)§	1–4	100	1,580	0.47	0.83
Rectum (154, except 154.0)	3, 4	101	784	0.44	0.80
Lung (162)	1, 4	499	920	0.83	0.99
Prostate (185)	1–4	300	1,380	0.78	0.99
Bladder (188)	1–4	300	1,380	0.78	0.99
Kidney (189)	1–4	100	1,580	0.47	0.83
Non-Hodgkin's lymphoma (200, 202)	1–4	117	1,563	0.51	0.87

* Each of the following sites had too few cases to analyze as a case group, but were included among "controls" for other sites: melanoma (n = 66), esophagus (n = 63), pancreas (n = 63), Hodgkin's (n = 31), liver (n = 30), testis (n = 9), penis (n = 9), gallbladder (n = 7), peritoneum (n = 7), myeloma (n = 7).

† For each case series, all other cancer patients interviewed served as controls, with the following exceptions: Lung cancer patients were excluded as controls for other sites. As controls for the rectal cancer series, only subjects interviewed in years 3 and 4 were included. As controls for the lung cancer series, only subjects interviewed in years 1 and 4 were included.

‡ Exposure prevalence refers to the percentage of the population exposed to the carcinogen. Power indicates the probability that such a risk will be detected with such sample sizes, such an exposure prevalence, a twofold risk and an alpha error of 0.05.

§ Includes sigmoid colon and rectosigmoid junction.

and a twofold risk, most of the sites included afford reasonable power. For the less common exposures, the probability of detecting twofold risks is over 0.7 only for four sites.

Defining exposure groups

The nine organic dusts were the exposure factors of interest. To estimate an odds ratio (OR) we must define case and control groups, which has been discussed above, and we must define what is meant by "exposed" versus "unexposed." Exposure status could be dichotomized in a variety of ways. While ever exposed/never exposed, as evaluated by our chemists, is the most straightforward, it runs the risk of diluting the "exposed" with subjects who had very low-level exposure or whose exposure status was questionable. It was decided to set up three exposure levels for each organic dust: "unexposed," "doubtful," and "substantial." At this stage, we wanted to distinguish, not so much between two levels of dosage, but rather between subjects who certainly had exposure above background levels and those who may not have. Available information on concentration, frequency, and reliability of the exposure inference was used for this purpose. Table 2 shows the criteria used to distinguish "doubtful" from "substantial" exposure. "Unexposed" meant never exposed at any level. "Doubtful" exposure includes those which were coded with low confidence or which were attributed at low concentration or frequency. Exposures which were of medium to high confidence *and* of medium to high concentration and frequency were considered to be "substantial."

TABLE 2

Criteria for designating "doubtful" and "substantial" exposure as a function of concentration, frequency, and reliability of exposure: study of associations of several cancer sites with nine organic dusts, Montreal, 1979–1983

Concentration × frequency†	Reliability*		
	Possible	Probable	Definite
≤3	Doubtful	Doubtful	Doubtful
4	Doubtful	Doubtful	Substantial
≥6	Doubtful	Substantial	Substantial

* Reliability refers to the coder's confidence that the exposure occurred.

† Concentration refers to the presumed level of exposure to the substance on a relative scale. Frequency refers to the proportion of the workweek during which exposure occurred. Each were scored on three-point scales, and thus the product can take the values 1, 2, 3, 4, 6, 8, and 9.

Methods of analysis

There was information on scores of nonoccupational factors and other occupational exposures besides the nine organic dusts. For each organic dust-cancer site association, potential confounders included the nonoccupational factors, the occupational exposures apart from the nine under study, and the other eight organic dusts. To reduce the problem to manageable dimensions, we carried out several stages of analysis.

We first selected certain potential confounders as "a priori" confounders to be included in every analysis. These were age, ethnic group, socioeconomic status as measured by self-reported income, cigarette smoking, and salubrity of the work history. This last variable was included because it is desirable that the case and control groups have similar job environments, except for the agent of interest. At this stage we tried to distinguish "clean" work histories from "dirty" ones. This was based on a rough impressionistic evaluation by our team of chemists of the salubrity of the job corresponding to each four-digit job category in the Canadian occupational classification system. Each job was scored from 0 to 8, and subjects' lifetime scores were computed by applying time-weighted averages to the job classes they had held in their lifetimes.

The first set of analyses to estimate odds ratios between the various sites and nine dusts was based on the procedure of Mantel and Haenszel (9). A priori stratification variables were age (35–54, 55–70 years), ethnicity (French, other), socioeconomic class (low, high), salubrity ("clean," "dirty"), cigarette consumption (none, low, high). Estimates were obtained both for ever exposed vs. never exposed and substantially exposed vs. never exposed. A flexible program was written for the purpose of computing large numbers of Mantel-Haenszel estimates in a single run (10).

Odds ratios that were suggestive of an association were subject to further refinement to take into account all other potential confounders. For each association between an exposure factor and a site of cancer earmarked for further investigation, the odds ratio, used as an index of association, was computed between the exposure factor and each of the hundreds of potential confounders (occupational and nonoccupational). We similarly computed the odds ratio between the site of cancer under study and the same list of potential confounders. Since a factor can confound an association if it is associated with the disease and with the exposure factor of interest, the overlap of these two sets of results provided us with a list of data-based potential confounders. To render operational these principles, an association was flagged either if the odds ratio exceeded 1.5 or was less than 0.67, or if the odds ratio was significantly different from 1.0 at the $p = 0.05$ level. Only those potential confounders flagged in both sets of analyses were retained.

The resulting list of confounders could then be included in a logistic regression analysis along with the exposure factor to provide estimates of the odds ratio and confidence limits, adjusted for the confounders. This was done using the GLIM program (11). Continuous variables were maintained as such; dummy variables were used for nominal or binary confounders. The organic dust(s) of interest for each site was (were) included as two dummy variables—one for "doubtful" exposure and one

for "substantial" exposure as defined in table 2.

RESULTS

Table 3 describes the exposure patterns of our entire study population (2,180 subjects) to each of the nine organic dusts. Wood dust is by far the most common exposure; 24.1 per cent of all subjects were considered to have had potential exposure to wood dust in at least one of their jobs. Most of these were considered definitely exposed (21.2 per cent of the entire sample). Only 12.5 per cent of the entire sample had been exposed at high frequency (more than 30 per cent of the day) and only 5.1 per cent were exposed at the high concentration level (on a relative scale). A large number, 9.8 per cent, had over 20 years exposure to wood dust in one form or another. Combining the various characteristics of exposure into a single index for the purpose of defining "substantial" versus "doubtful" exposure as described above, we find that of all subjects 15.8 per cent were in the "substantial" exposure group for wood dust. By contrast, fur dust is the least common, with a lifetime work prevalence of 2.7 per cent for any exposure and 2.2 per cent for "substantial" exposure. Table 4 shows for each dust the main occupation groups in which it occurred in our population. Most of these substances occurred in many job classes apart from those shown in the table. Also, the indication that a substance was attributed to some workers bearing a given job title does not imply that this exposure was attributed to all workers with that job title. For instance, while many of those exposed to grain dust were "materials handlers," only a fraction of "materials handlers" were exposed to grain dust. As expected, exposure to fabrics, synthetic fibers, cotton, and wool occurred in many of the same occupations.

Table 5 shows the odds ratios, confidence limits, and numbers of exposed cases for each of $7 \times 9 = 63$ disease-exposure associations when using a Mantel-Haenszel procedure stratifying on age, cigarette smoking, socioeconomic status, ethnicity, and salubrity of the job history. This table includes the site category colorectal as a case series, but not its subsites. The odds ratios refer to the risk of "substantial" exposure vis-à-vis "unexposed." Five odds ratios were significantly elevated at the two-sided $p = 0.05$ level, namely, prostate-grain dust, prostate-paper dust, bladder-synthetic fibers, colorectal-grain dust, and stomach-wood dust. There are several ways to estimate how many statistically significant results would be expected on the global null hypothesis of no association (12). One way is to assume that 0.025 of all associations should be significantly higher than

TABLE 3

Percentage of all 2,180 subjects exposed to each of nine organic dusts according to degree of exposure: study of associations of several cancer sites with nine organic dusts, Montreal, 1979–1983

Substance	Any exposure*: %	High reliability: %	High frequency: %	High concentration: %	>20 years exposure: %	"Substantial" exposure†: %
Wood dust	24.1	21.2	12.5	5.1	9.8	15.8
Paper dust	7.0	5.7	4.9	0.5	2.3	3.8
Grain dust	7.1	5.9	1.2	3.2	2.2	4.2
Flour dust	3.9	3.3	2.6	1.2	1.4	3.2
Fabrics dust	9.4	8.4	6.3	1.7	4.2	6.3
Cotton dust	7.4	6.8	5.6	2.4	2.8	6.0
Wool dust	5.2	4.3	3.5	1.3	2.3	3.9
Synthetic fibers	5.1	4.2	3.4	1.7	2.2	3.4
Fur dust	2.7	2.3	1.8	1.0	1.0	2.2

* Exposure attributed with any degree of reliability and at any frequency, concentration and duration.
† As defined in table 2.

TABLE 4

The main occupations in which each organic dust was attributed in the entire study group: study of associations of several cancer sites with nine organic dusts, Montreal, 1979–1983

Dust	n*	Main occupations in which it was coded (% of n who were in this occupation)
Wood	525	Construction trades—carpenters, painters, pipefitters, plumbers, etc. (30.3); forestry and logging occupations (13.3); fabricating and assembling wood products (8.8).
Paper	153	Material handling (19.0); printing occupations (16.3); mail carriers, clerks, supervisors (9.8); shipping, receiving, stock clerks (9.2); paper product fabricating (8.5).
Grain	156	Farming, horticultural, animal husbandry occupations (58.3); material handlers (14.1); farm management (9.6).
Flour	85	Food processing occupations (35.3); material handlers (18.8); chefs, cooks, and related food handlers (15.3).
Fabrics	205	Fabricating, assembling, repairing occupations—textile, fur, and leather products (30.7); laundering, pressing, other apparel and furnishings service occupations (12.2); janitors, cleaners (8.3); sales occupations (8.3); material handlers (7.8); shipping, receiving, stock clerks (6.8).
Cotton	161	Fabricating, assembling, repairing occupations—textile, fur, and leather products (32.9); textile processing (17.4); laundering, pressing, other apparel and furnishing services occupations (6.8); shipping and receiving clerks, stock clerks (6.8); mechanics and repairmen of industrial machinery (6.8).
Wool	112	Fabricating, assembling repairing occupations—textile, fur, and leather products (44.6); textile processing (14.3); pressing, laundering, etc. (8.9); mechanics and repairmen of industrial machinery (6.2).
Synthetic fibers	113	Fabricating, assembling, repairing occupations—textile, fur and leather products (36.3); textile processing occupations (15.0); laundering, dry cleaning, pressing (8.8); mechanics and repairmen of industrial machinery (7.1).
Fur	60	Furriers, milliners, other fabricating and repairing occupations—fur, leather products (36.7); hide and pelt processing (18.3).

* This is the number of persons exposed at any level (i.e., "doubtful" plus "substantial"); *n* is the denominator for each percentage corresponding to this dust.

1.00, i.e., $0.025 \times 63 = 1.6$. Another is to assume the distribution of log odds ratios would be symmetric around 1.00, in which case the number of significantly low odds ratios, namely two, is an estimate of the expected number of significantly high odds ratios. It appears that there are more significiantly high odds ratios than expected

TABLE 5

Odds ratios between seven sites of cancer and exposure at the "substantial" level to each of nine organic dusts, based on Mantel-Haenszel analyses with five stratifying variables†: study of associations of several cancer sites with nine organic dusts, Montreal, 1979–1983*

Site	Wood			Paper			Grain			Flour			Fabrics		
	OR‡	95% CI	n	OR	95% CI	n	OR	95% CI	n	OR	95% CI	n	OR	95% CI	n
Stomach	1.7	1.1–2.8	35	1.1	0.4–3.2	6	0.7	0.3–1.8	5	0.8	0.3–2.3	4	0.9	0.4–1.6	9
Colorectal	1.2	0.9–1.7	66	0.8	0.4–1.7	11	1.7	1.1–2.7	25	1.2	0.6–2.2	15	1.0	0.6–1.6	26
Lung	1.3	1.0–1.8	98	1.4	0.7–2.9	23	0.6	0.4–1.1	18	1.0	0.6–2.0	18	0.9	0.5–1.6	27
Prostate	1.1	0.7–1.5	45	1.9	1.0–3.3	16	1.9	1.0–3.8	17	1.2	0.6–2.5	10	1.1	0.6–1.9	18
Bladder	0.9	0.6–1.2	39	0.5	0.2–1.4	6	0.6	0.3–1.2	8	0.5	0.2–1.3	5	1.3	0.8–2.0	22
Kidney	1.0	0.5–1.8	13	0.9	0.3–2.8	3	0.9	0.3–2.2	4	1.8	0.6–4.8	5	0.9	0.4–2.0	7
Non-Hodgkin's lymphoma	0.5	0.2–0.9	11	0.7	0.2–1.8	3	0.5	0.1–1.7	2	0.3	0.0–1.3	1	0.9	0.4–1.9	7

* See table 2 for definition of "substantial" exposure.
† Stratifying variables: age, socioeconomic status, ethnic group, cigarette smoking and salubrity of job history.
‡ OR, odds ratio; 95% CI, 95% confidence interval; *n*, number of exposed cases at "substantial" level.

on the global null hypothesis. In addition to the five associations that were significant at the 0.05 level, we also earmarked those which were of borderline significance in table 5 or which were significant in a separate analysis that compared all exposed (i.e., "doubtful" plus "substantial") with unexposed. Thus were added the following associations: lung-wood, colorectal-wood, colorectal-synthetics, lymphoma-cotton.

Although not shown in table 5, analogous analyses were carried out on colon, rectosigmoid and rectum separately. Using the same criteria, the following associations were earmarked: rectum-fabrics, rectum-wool, and colon-grain. The colon-grain association was in fact entirely responsible for the significant colorectal-grain association; therefore only the colon-grain association will be examined in depth.

Thus, 11 associations were selected for in-depth analysis. For each of these, a series of analyses was carried out, as described in the Methods section, to identify a short list of potential confounders among the hundreds of occupational and nonoccupational variables available. A logistic regression analysis was then carried out for each association. When a potential risk factor was apparently associated with two or more different sites (e.g., grain with prostate and colon), we excluded from the control group for each of the sites the other site(s) associated with the same exposure.

Table 6 shows, for each of the 11 selected associations, the odds ratios corresponding to "doubtful" and "substantial" exposure and to "substantial" exposure for ≤15 or >15 years. A few of the odds ratios in the "substantial" exposure column have changed from the preceding Mantel-Haenszel analysis. The wool and fabric dust associations with rectum cancer fell towards the null value and were no longer even of borderline significance. The remaining nine associations were either significant or barely nonsignificant at $p = 0.05$ for subjects "substantially" exposed. The stomach-wood and colorectal-synthetic fiber associations were also significant at the "doubtful" exposure level.

The associations that were statistically significant and manifested greatest risk among those with longest exposure were lung-wood dust, stomach-wood dust, lymphoma-cotton dust, bladder-synthetic fibers, and colorectal-synthetic fibers. The following associations were statistically significant at one level or another but did not manifest a "dose-response" relation with duration: prostate-paper dust, prostate-grain dust, and colon-grain dust. The colorectal-wood dust association was barely nonsignificant, but there was a hint of a "dose-response" with duration.

To determine for each association whether the elevated risk was concentrated in a particular occupation, or was spread

TABLE 5—*Continued*

Site	Cotton			Wool			Synthetic fibers			Fur		
	OR	95% CI	*n*	OR	95% CI	*n*	OR	95% CI	*n*	OR	95% CI	*n*
Stomach	0.4	0.2–1.0	5	0.9	0.4–1.9	7	0.8	0.3–2.0	6	0.8	0.2–3.1	3
Colorectal	1.2	0.7–2.1	30	1.0	0.5–1.8	17	1.5	0.8–2.9	22	0.7	0.3–1.7	7
Lung	0.8	0.4–1.3	25	0.5	0.3–1.0	15	0.5	0.2–1.1	11	0.8	0.4–1.7	9
Prostate	1.0	0.6–1.8	16	0.9	0.4–1.8	9	0.7	0.3–1.7	7	1.1	0.5–2.4	9
Bladder	1.2	0.7–2.1	21	1.3	0.7–2.4	15	1.6	0.9–3.0	17	0.8	0.3–1.8	5
Kidney	1.1	0.5–2.7	7	1.0	0.3–3.0	5	0.8	0.2–2.8	4	2.4	0.7–7.6	5
Non-Hodgkin's lymphoma	1.6	0.9–2.8	11	1.0	0.4–2.6	5	0.7	0.2–2.2	4	1.3	0.4–4.8	3

TABLE 6

Odds ratios at various levels and durations of exposure, for 11 selected associations: study of associations of several cancer sites with nine organic dusts, Montreal, 1979–1983*

Association		"Doubtful" exposure†			"Substantial" exposure†			"Substantial" exposure†					
								≤15 years exposure			>15 years exposure		
Substance	Site	OR‡	95% CI	*n*	OR	95% CI	*n*	OR	95% CI	*n*	OR	95% CI	*n*
Wood dust	Lung	1.1	0.7–1.7	46	1.5	1.0–2.1	98	1.3	0.8–2.2	52	1.7	1.0–2.7	46
Wood dust	Stomach	1.7	1.0–2.9	20	1.5	1.0–2.4	35	1.2	0.6–2.2	17	1.9	1.1–3.3	18
Wood dust	Colorectal	1.0	0.6–1.5	31	1.2	0.9–1.7	66	1.1	0.7–1.7	32	1.4	0.9–2.2	34
Paper dust	Prostate	0.9	0.4–1.9	8	2.0	1.0–3.8	17	2.8	1.2–6.3	12	1.1	0.4–3.4	4
Grain dust	Prostate	1.3	0.7–2.8	12	2.2	1.2–4.2	19	3.3	1.3–8.3	10	1.5	0.6–3.7	7
Grain dust	Colon	0.8	0.3–2.0	5	2.6	1.4–4.9	17	3.6	1.5–8.9	9	1.9	0.8–4.6	8
Fabric dust	Rectum	0.2	0.0–1.3	2	1.0	0.4–2.7	10	0.8	0.2–3.3	3	1.2	0.3–3.9	7
Cotton dust	Lymphoma (non-Hodgkin's)	0.7	0.1–5.0	1	1.9	1.0–3.7	11	1.2	0.4–3.3	4	3.0	1.3–7.2	7
Wool fibers	Rectum	3.7	0.8–17.1	3	2.0	0.7–5.0	8	1.1	0.2–12.3	3	2.1	0.6–7.4	5
Synthetic fibers	Colorectal	3.3	1.3–8.2	12	1.5	0.9–2.7	22	0.7	0.3–1.7	6	3.0	1.4–6.6	16
Synthetic fibers	Bladder	1.4	0.5–3.9	5	1.8	1.0–3.2	17	1.5	0.6–3.5	7	2.1	1.0–4.4	10

* Based on logistic regression. There was a distinct regression model for each association. The list of variables included in each model was a subset of the entire set of potential confounders and was selected according to the procedure described in the text. For each association the control group is the same as that used for table 5, excluding any other sites which are presented in this table in association with the same exposure factor.

† As defined in table 2.

‡ OR, odds ratio; 95% CI, 95% confidence interval; *n*, number of cases exposed at this level. For each association an odds ratio of 1.0 is implicit for the unexposed.

among all groups exposed to the substance, we examined the occupation distribution of the high-risk site(s) for each high-risk organic dust. For stomach cancer, the excess risk among wood-exposed workers was concentrated in forestry and logging. The odds ratio for stomach cancer among wood-exposed forestry and logging workers was 4.7 (95 per cent confidence interval (CI): 1.5–14.1) for those with "doubtful" exposure, and 1.7 (95 per cent CI: 0.6–5.1) for those with "substantial" exposure. For lymphoma cases exposed to cotton dust, most were in textile processing. The odds ratio for lymphoma among textile processing workers with "substantial" exposure to cotton dust was 12.6 (95 per cent CI: 4.7–33.3). Other workers exposed to cotton dust had no excess risk. About one-third of the prostate cancer cases exposed to grain dust were "materials handlers," mainly grain elevator operators. The odds ratio for prostate cancer among materials handlers exposed to grain dust was 10.5 (95 per cent CI: 1.8–60.0) in the "doubtful" exposure group and 3.2 (95 per cent CI: 0.8–12.5) in the "substantial" exposure group. There was a 1.6 odds ratio among other workers exposed to grain dust at the "substantial" level. For each of the remaining associations the high odds ratios were not concentrated in any single occupation.

To appreciate the potential public health importance of each of these associations, and thus the relative priority for corroborative research, we estimated for each association the population attributable risk per cent using Miettinen's procedure (13), assuming the risk to be limited to workers with "substantial" exposure to the substance. Multiplying the site-specific attributable risk per cent by the proportion of all cancers that this site represents, it was possible to estimate the per cent of all cancers that may be attributable to this association. Thus 1.4 per cent of all cancers among males aged 35–70 years were estimated to be attributable to the lung cancer-wood dust association. For the other eight significant or borderline significant associ-

ations, the analogous percentages varied from 0.2 to 0.4.

Discussion

When our project was designed, none of the nine substances in the present report were thought to be risk factors for the sites of cancer that we were studying. The nine substances were chosen for analysis and presentation at this time because they had a similar chemical/physical characteristic and because they were relatively common exposures.

Wood dust

Wood dust has been recognized as a risk factor for nasal-sinus cancer (5). There have been uncorroborated reports of excess Hodgkin's disease among woodworkers (14, 15). Dubrow and Wegman (16), in a synthesis of 12 surveillance studies based on death certificates and tumor registers, reported a small but consistent excess of lung cancer among carpenters and among construction workers. Recently there have been case-control studies implicating wood dust as a risk factor for lung cancer in a town in Sweden (17), in coastal Georgia (18), and in northern Florida (19). Excess lung cancer risk among carpenters in the United States was found in a large prospective cohort study (20) and in a retrospective cohort study (21). On the other hand, in large well-conducted retrospective cohort studies of furniture workers in England (22) and carpenters in Denmark (23), no excess risks of lung cancer were found. Our finding of an excess of lung cancer among wood-exposed workers not only supports the various positive findings but is more persuasive than most because we were able to control for smoking and other factors including other occupational exposures (e.g., asbestos) and we have more reliable information on the wood exposure than did many of the other studies. Confounding by some occupational exposures that we did not include in our study, or by an exposure that was included but poorly coded, is possible. Wood dust is a very general designation; it may very well be that the apparently conflicting results from different cohorts are due to different types of wood dust exposure in different places. Based on the Montreal experience, the odds ratio for lung cancer is not high; however, the number of persons exposed to this substance is very high and thus the population attributable risk is considerable.

Dubrow and Wegman (16) also reported a small but consistent excess of stomach cancer among carpenters and cabinet makers in the various routine record surveillance studies. Stellman and Garfinkel (20) found a significantly high stomach cancer rate among carpenters and joiners in a national US prospective cohort study as did Milham (21) in a retrospective cohort study. Milham and Demers (24) found a high stomach cancer proportionate mortality ratio among pulp mill workers. In the British cohort of woodworkers who had no excess of lung cancer, there was a hint of an excess stomach cancer risk (22). Curiously, the risk was lower for the most highly exposed workers. We found slightly lower risk of stomach cancer among those with "substantial" exposure compared with those with "doubtful" exposure, although both were significant. The risk was greatest among those with longer exposure. The concentration of the excess risk among loggers and foresters has no obvious explanation if the risk is due to wood dust itself. The evidence for a wood-colorectal association is weak and is not supported by previous studies.

The possible carcinogenicity of wood dust for such diverse sites as nasal sinus, lung, and stomach is biologically plausible since the dust may contact each of those organs. It has been postulated that coal dust exposure may increase risks of lung cancer in some workers and stomach cancer in others, depending on the subject's respiratory function (25, 26). This hypothesis may be just as relevant for wood dust exposure. The International Agency for Research on Cancer (5) has summarized some of the chemical, physical, and biologic

agents which may accompany and confound wood dust exposure. If the associations are real, there would remain crucial questions about the types and forms of wood dust that are dangerous, and about latency and interactions. In any case, wood dust, one of the oldest occupational exposures, may be a major human carcinogen.

Grain dust

No previous studies have focussed on grain dust as a risk factor for cancer. Three of the routine record surveillance studies evaluated by Dubrow and Wegman (16) and a death certificate study in Iowa (27) showed slight excess risks of prostate cancer among farmers, who constitute one of the main groups exposed to grain dust. In Washington State, Milham (28) found excess prostate cancer specifically among wheat farmers and grain elevator operators. It is of interest that the risk of prostate cancer in our study was even higher among grain-exposed materials handlers, mainly grain elevator workers, than among grain-exposed farmers. Exposure to grain dust is higher among the former. Excess colon cancer risk among grain-exposed workers has not been reported previously. Neither of the grain-related associations in our data displays the "dose-response" relation with duration of exposure that would be expected with a causal agent; confidence limits, however, were quite wide. Like any other associations in our data, the possibility of confounding by another factor must be considered. Preservatives form a class that are of particular interest with respect to grain dust since we have not coded preservatives and thus could not control for them in analysis. Ethylene dibromide is one of the main fumigants and has been shown to be a mutagen and carcinogenic to animals (29).

Organic textile dusts

Wool dust, fur dust, and fabric dust were not significantly associated with any cancer sites, once the statistical control for potential confounders was complete. However, excess risks of non-Hodgkin's lymphoma among workers exposed to cotton dust and excess risks of bladder and colorectal cancers among workers exposed to synthetic fibers cannot be dismissed.

Like most other job titles the term "textile worker" can cover a great variety of occupational environments. In their synthesis of 12 surveillance studies, Dubrow and Wegman (16) reported that textile workers showed slight excess risk for cancer of the mouth, but not for any of the sites covered by our study. However, one of the studies reported an excess of lymphomas among textile workers (29). In retrospective cohort mortality studies among male cotton mill workers in Georgia, North Carolina, and Lancashire, no excess cancer risks were found; however, the numbers of expected cases were so small that twofold risks of cancer of specific sites would not likely have been detected (30–32). In a large study of death certificates of females in North Carolina, elevated proportionate mortality ratios were found among textile workers for a few sites of cancer including non-Hodgkin's lymphoma (33). Our finding of a significant excess of non-Hodgkin's lymphoma among males with cotton exposure was concentrated among textile producers. Although the agent or mechanism of carcinogenesis is not obvious, the association between non-Hodgkin's lymphoma and cotton dust merits attention.

The surveillance studies reviewed by Dubrow and Wegman (16) did not report excess risks of bladder cancer or colorectal cancer among textile workers. However, case-control studies of bladder cancer in Britain (34) and in New Jersey (35) did show excess risks among textile workers; those authors suggested that carcinogenic textile dyes may have been responsible for their findings. There was no indication as to the types of textiles or the types of dyes such workers may have been exposed to. In our study there was no association between bladder or colorectal cancer and cotton, wool or fabrics dust. Only synthetic fiber exposure showed risks. It should be reem-

phasized that the exposure examined here was the dust or fiber. Thus, for example, workers involved in the production of synthetic fibers but not exposed to the end product in the form of a dust or fiber, might not have had the exposure attributed. We coded the general categories "organic dyes and pigments" and "inorganic dyes and pigments"; in our data there was no confounding by these exposures. If the associations between bladder and colorectal cancers and synthetic fibers are not statistical false positive results, it suggests three possible hypotheses: a) that the specific types of dyes used with synthetic fabrics in the Montreal area are bladder and/or colorectal carcinogens whereas those used with other textiles are not, b) that the same dyes may be used with the various textiles but that synthetic fibers permit greater leaching of the dyes once the fibers are ingested, or c) that one or more of the types of synthetic fiber is itself decomposed and metabolized into a bladder or colorectal carcinogen. The principal types of synthetics in our study population would probably be polyester, acrylic, nylon, and acetate. In due course we will try to determine whether the risks can be identified with a particular fiber type. It is of particular interest that Vobecky et al. (36) have reported an excess of colorectal cancer in a synthetic fiber production plant.

General comments and caveats

Our statistical analysis strategy had the effect of adjusting each estimate of odds ratio for hundreds of occupational and nonoccupational factors. Although the final logistic regression model may have included only a handful of factors, the preceding steps of our analysis effectively ruled out the risk of serious confounding due to any other of the variables at our disposal. Of course, confounding could still occur if those variables had not been accurately ascertained.

No single epidemiologic study can provide conclusive evidence of an association. A result observed in an hypothesis-generating exercise, where the association had very low prior probability, would lead to a more tentative conclusion than one coming out of a study designed to test a hypothesis which already had some previous support. The essential point is that it takes several pieces of corroborative evidence before an association can be accepted as causal. Whether a given piece of evidence comes from an hypothesis-generating or an hypothesis-testing study per se is irrelevant. It is the sum of the evidence that counts.

This study has limitations. The validity of the exposure coding was not perfect; such errors could lead to false negative results if one of the organic dusts under study were seriously miscoded or it could lead to false positive results if an important confounding factor were seriously miscoded. The exposure checklist is finite; it is conceivable that a potential confounder may have been omitted. Also, any "positive" statistical finding has some chance, alpha, of being a false positive. On the other hand, the power to detect risks was only moderate for most of the sites analyzed; thus the absence of a significant association may not in itself be persuasive. As larger sample sizes are accumulated, analyses will be redone to provide stronger evidence of the harmlessness of substances not associated with cancer.

From a methodological viewpoint, it is instructive that we were able to detect distinct patterns of risk associated with exposure to fabric dust, wool dust, cotton dust, and synthetic fibers, whereas studies based on job titles might lump all people exposed to these substances together as textile workers.

Our purpose was to generate hypotheses. Nine associations seem noteworthy—namely, wood dust with lung, stomach and colorectal cancers; grain dust with prostate and colon cancers; paper dust with prostate cancer; synthetic fibers with bladder cancer and colorectal cancer; and cotton dust with non-Hodgkin's lymphoma. Of these, the associations with grain dust and paper dust did not show evidence of dose-response and the wood-colorectal association was weak.

All of these associations will be under continued scrutiny in our own growing data base. Each association must be considered on its merits. The fact that some of them have not previously been reported may reflect the lack of sensitivity of most previous surveillance approaches rather than meaningful contradictions between our study and others. Because the assessment of exposure was much more precise than any previous surveillance attempt (most others were based on the last job title a person had, as noted on his death certificate), because the disease ascertainment was based on histologic confirmation (rather than the error-prone death certificate cause), and because a comprehensive list of potential confounders, occupational and nonoccupational, were taken into account (unlike most surveillance studies which have only taken age and sex into account), the individual results of this surveillance study must be given greater credence than those of most others.

References

1. US National Research Council. Toxicity testing: strategies to determine needs and priorities. Washington, DC: National Academy Press, 1984.
2. Siemiatycki J, Day N, Fabry J, et al. Discovering carcinogens in the occupational environment: a novel epidemiologic approach. JNCI 1981;66:217–25.
3. Hoar SK, Morrison AS, Cole P, et al. An occupation and exposure linkage system for the study of occupational carcinogenesis. J Occup Med 1980;22:722–6.
4. Coggon D, Pannett B, Acheson ED. Screening for new occupational hazards of cancer in young persons. Ann Occup Hyg 1984;28:145–50.
5. IARC monographs of the evaluation of the carcinogenic risk of chemicals to humans. Vol. 25. Wood, leather and some associated industries. Lyon: IARC, 1981.
6. Siemiatycki J, Gérin M, Richardson L, et al. Preliminary report of an exposure-based, case-control monitoring system for discovering occupational carcinogens. Teratogenesis Carcinog Mutagen 1982;2:169–77.
7. Gérin M, Siemiatycki J, Kemper H, et al. Obtaining occupation exposure histories in epidemiologic case-control studies. J Occup Med 1985;27:420–6.
8. Richardson L, Siemiatycki J. Case-control study methods—when to interview subjects and nonresponse bias. Presented at Fourth International Symposium on Epidemiology in Occupational Health, Como, Italy, 1985.
9. Mantel N, Haenszel W. Statistical aspects of the analysis of data from retrospective studies of disease. JNCI 1959;22:719–48.
10. Dewar RAD, Siemiatycki J. A program for point and interval calculation of odds ratios and attributable risks from unmatched case-control data. Int J Biomed Comput 1985;16:183–90.
11. Baker RJ, Nelder JA. The GLIM System. Release 3. Generalised linear interactive modelling. Oxford: Royal Statistical Society, 1978.
12. Thomas DC, Siemiatycki J, Dewar R, et al. The problem of multiple inference in studies designed to generate hypotheses. Am J Epidemiol 1985;122:1080–95.
13. Miettinen OS. Proportion of disease caused or prevented by a given exposure, trait or intervention. Am J Epidemiol 1974;99:325–32.
14. Milham S Jr. Occupational mortality in Washington State: 1950–1971. Washington, DC: DHEW Publication no. (NIOSH) 76-175-A, 1976.
15. Petersen GR, Milham S Jr. Hodgkin's disease mortality and occupational exposure to wood. JNCI 1974;53:957–8.
16. Dubrow R, Wegman D. Setting priorities for occupational cancer research and control-synthesis of the results of occupational disease surveillance studies. JNCI 1983;71:1123–42.
17. Esping B, Axelson O. A pilot study on respiratory and digestive tract cancer among woodworkers. Scand J Work Environ Health 1980;6:201–5.
18. Harrington JM, Blot WJ, Hoover RN, et al. Lung cancer in coastal Georgia: a death certificate analysis of occupation. JNCI 1978;60:295–8.
19. Blot WJ, Davies JE, Brown LM, et al. Occupation and the high risk of lung cancer in northeast Florida. Cancer 1982;50:364–71.
20. Stellman SD, Garfinkel L. Cancer mortality among woodworkers. Am J Ind Med 1984;5:343–57.
21. Milham S Jr. A study of the mortality experience of the AFL-CIO United Brotherhood of Carpenters and Joiners of America, 1969–1970. DHEW publication no. (NIOSH) 74-129. Springfield, VA: National Technical Information Service, 1974.
22. Acheson ED, Pippard EC, Winter PD. Mortality of English furniture makers. Scand J Work Environ Health 1984;10:211–17.
23. Olsen J, Sabroe S. A follow-up study of nonretired and retired members of the Danish carpenter/cabinet maker's trade union. Int J Epidemiol 1979;8:375–82.
24. Milham S Jr, Demers RY. Mortality among pulp and paper workers. J Occup Med 1984;26:844–6.
25. Meyer MB, Luk GD, Sotelo JM, et al. Hypothesis—the role of the lung in stomach carcinogensis. Am Rev Respir Dis 1980;121:887–92.
26. Gamble JF, Ames RG. The role of the lung in stomach carcinogenesis—a revision of the Meyer hypothesis. Med Hypotheses 1983;11:359–64.
27. Burmeister LF, Everett GD, Van Lier SF, et al. Selected cancer mortality and farm practices in Iowa. Am J Epidemiol 1983;118:72–7.
28. Milham S Jr. Occupational mortality in Washington State: 1950–1971. DHEW publication no. (NIOSH) 76-175-A. Washington, DC: US GPO, 1976.

29. Decoufle P, Stanislawczyk K, Houten L, et al. A retrospective survey of cancer in relation to occupation. DHEW publication no. (NIOSH) 77-178. Washington, DC: US GPO, 1977.
30. Henderson V, Enterline P. An unusual mortality experience in cotton textile workers. J Occup Med 1973;15:717–19.
31. Berry G, Molyneux MKB. A mortality study of workers in Lancashire cotton mills. Chest 1981;79:(Suppl)115–55.
32. Merchant J, Ortmeyer C. Mortality of employees in two cotton mills in North Carolina. Chest 1981;79:(Suppl)65–105.
33. Delzell E, Grufferman S. Cancer and other causes of death among female textile workers, 1976–78. JNCI 1983;71:735–40.
34. Anthony H, Thomas G. Tumors of the urinary bladder—an analysis of the occupations in 1,030 patients in Leeds, England. JNCI 1970;45:879–95.
35. Schoenberg J, Stemhagen A, Mogielnicki A, et al. Case-control study of bladder cancer in New Jersey. 1. Occupational exposures in white males. JNCI 1984;72:973–81.
36. Vobecky J, Devroede G, Caro J. Risk of large-bowel cancer in synthetic fiber manufacture. Cancer 1984;54:2537–42.